treated in the open air with only the face exposed as compared with counts in the ward.

2. In the majority of patients with fever and leukocytosis there is a small percentage decrease in the total leukocytes when treated in the open air with only the face exposed as compared with the count when treated in the ward. Under like conditions there is no constant change in the polymorphonuclear counts.

These experiments were carried out at the House of Relief in the service of Dr. T. B. Barringer, to whom I am much indebted for advice and assistance in the preparation of this paper.

FATAL PNEUMOTHORAX FOLLOWING EXPLORATORY PUNCTURE.[1]

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In a paper on Accidents and Deaths from Exploratory Puncture of the Pleura,[2] the statement was made that the importance of pneumothorax after exploratory puncture depended upon the condition of the patient at the time of its occurrence and upon the physical state of the perforated lung, which might permit contraction of the puncture or favor extreme or progressive pneumothorax. This was based entirely upon a study of cases in the literature as no instances of this accident were found in the records of the New York Hospital during the twelve and a half years under consideration. Since then 2 fatal cases of this character have forced the conclusion that the physical state of the opposite lung may be of even greater importance.

Case No. 175,676, a widow, aged fifty-eight years, was admitted to New York Hospital on February 7, 1912, complaining of pain in the chest and cough. The only significant portion of her previous history was that of chronic cough for many years. In February, 1911, she was treated in the New York Hospital for an attack which was diagnosticated as pneumonia and resembled it in history, blood findings, and course, though the sputum was never blood-tinged. Cough continued until the present illness. About December 29, 1911, she had cough, chills, and fever every night, moderate sputum, never blood-tinged, and pain in both chests increased by coughing. Since January 4 she had been confined to bed and had lost considerable weight. On February 7, examination showed emaciation. Over both lungs there were slightly woody resonance, high-pitched voice, and occasional sibilant and subcrepitant rales. Posteriorly, over the left base, where signs of consolidation had been present a year before, were impaired resonance and diminished breathing. The sputum was scanty, thick, mucopurulent. Six specimens were negative for tubercle bacilli. The temperature usually ranged between 98° F. and 101° F. or less each day, with respirations at first 36 to 46, later 22 to 26, and pulse 92 to 104. The urine was that of chronic interstitial nephritis. The leukocytes numbered about 10,000, of which about 70 per cent. were polymorphonuclears. On February 22 the left chest posteriorly from the seventh rib to the base and extending out four inches from the median line showed dulness and many subcrepitant rales; over the upper part of this area were bronchial breathing, voice, and whisper.

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[1] Read before the New York Academy of Medicine, Section on Medicine, May 21, 1912

[2] Surg., Gyn., and Obstet., 1911, xiii, 607.

Two days later I noted in the seventh and eighth spaces, about two to two and a half inches from the median line, a small area over which cavernous breathing and whisper were heard only after productive cough. After unproductive cough there were heard coarse, almost musical moist rales. Within a few minutes after coughing the signs suggestive of a cavity would disappear completely excepting occasional rales. Below the above area were dulness, diminished breath and voice sounds to the base of the lung. The patient's general condition improved and she was allowed to sit up. On the morning of March 5, she did not feel well and had some dyspnea. At 8 A.M. the temperature was 102°; respiration, 24; pulse, 100. Physical signs were unchanged. Aspiration of the right chest was ordered. Hearing of this, I reached the ward just as a needle had been inserted, at 4.45 P.M., in the sixth right intercostal space just internal to the vertebral border of the scapula. The needle was carefully inserted about one inch and nothing obtained. The patient complained, immediately after its withdrawal, of intense pain at the site of puncture and in the front of the right chest, then showed severe and increasing dyspnea and cyanosis. Signs of subcutaneous emphysema appeared over an area three or four inches in diameter around the site of puncture. Over the entire right chest were dull tympany and very distant breath sounds. The right border of cardiac dulness was displaced from the right to the left sternal margin. A diffuse apex-beat was indistinctly felt in the fifth space 13 cm. out. At 4.55 P.M., systolic blood pressure was 90, pulse 108, feeble and irregular in frequency. Magendie's solution, caffeine sodiosalicylate, and adrenalin were administered, but the patient became more cyanotic and was unconscious by 5 P.M., when blood pressure was only 60, pulse 120. The note over the right chest gradually became more dull and lost its tympanitic quality. Through a needle inserted in the third right interspace in the midclavicular line air was expressed during expiration, apparently not under good pressure. Death occurred at about 5.10.

Autopsy by Dr. R. G. Stillman showed the diaphragm extending to the fifth interspace on the right side and to the fourth on the left. The right lung seemed collapsed in part and the heart was displaced to the left. The right pleural cavity was dry and was filled over the apex of the upper lobe of the lung with dense fibrous adhesions. The puncture wound in the sixth right intercostal space, 5 cm. from the median line, occupied a position where the pleura was free from adhesions and corresponding to the apex of the lower lobe. The right upper lobe was somewhat lacerated by its removal but otherwise appeared normal. The surface of the lower lobe was smooth, glistening, and grayish-pink. No evidence of a puncture of the lung was found. A few encapsulated fibrous nodules were scattered through this entire lung. Two or three of these were calcareous. The left pleural cavity was entirely obliterated by firm adhesions. The left lung on section presented a dark-red surface from which on pressure a moderate amount of red, frothy fluid exuded. Along the posterior border of the lower lobe was an area of indurated tissue, about 4 by 9 cm., in which could be seen a multilocular cavity occupying almost all of this portion of the lung. The lining of this cavity was smooth. Opening into it were two or more dilated bronchioles. The bronchial mucous membrane was deeply injected and covered with mucopus. The walls of the bronchi appeared to contain an excessive amount of cartilage which in many places exhibited calcareous infiltration. Besides these signs of adhesive pleurisy at the right apex and right pneumothorax, with chronic pleurisy and chronic fibroid phthisis with cavity formation in the left lung, other lesions included those of chronic interstitial nephritis, chronic interstitial splenitis, perihepatitis, fibrous goitre, and a calcified uterine fibroma.

Of the possible causes of pneumothorax, entrance of air through the needle can be positively excluded, as the needle was attached, before introduction, to a Connell vacuum bottle with active suction. That air could not have entered subsequently through the puncture wound in the chest-wall was shown by the presence of a drop of blood over the wound until it was closed with collodion. That it was not the result of laceration of the adherent right apex was shown by the absence of signs of antemortem hemorrhage in any of the lacerations produced during removal of the lung at autopsy. Although no puncture hole could be found in the lung, the occurrence of a small perforation by the needle seems proved by exclusion. Operation upon the chest would have been ineffective because the asphyxia was due to the collapse of the only serviceable lung.

The chief object in reporting this case is to emphasize the statement made in my previous paper[3] that the greatest danger exists in puncturing cases in which chronic inflammatory processes are present. That assertion was made with reference to the danger of causing hemorrhage or pleural reflexes when puncturing the diseased lung. As regards the production of pneumothorax, some claim that it is most likely to occur when pleural adhesions are present or consolidation of the lung prevents closure of the puncture. Others think it occurs most often when the pleura is normal and the lung diseased, especially when there is softening in the lung near the pleural surface but not involving it. In the case which I have reported and one to which I shall refer briefly, the punctured lung tissue was absolutely or practically normal.

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[3] Loc cit.

When the patient's general condition is not too poor, when the perforated lung is in such physical condition as to permit closure of the puncture and so prevent extreme and progressive pneumothorax; and when the opposite lung is in good condition, unilateral pneumothorax may pass almost unobserved. In a patient (28 58, 486) who died in the New York Hospital in 1897 of acute pancreatitis after his right chest had been repeatedly explored, the autopsy showed pneumothorax of the right pleural cavity. The right lung was collapsed and inflation under water showed two small punctures, but the lungs were otherwise normal. In this case there were no subjective symptoms of pneumothorax.

A second case of fatal pneumothorax following exploratory puncture was a longshoreman, aged thirty-nine years, who was admitted to the Medical Division (No. 181442) on February 29, 1912, apparently suffering from lobar pneumonia involving the right lower lobe. Fever continued and signs of consolidation of the right lower lobe and the apex of the upper lobe persisted, especially near the spine of the scapula, where they suggested an interlobar empyema. Two punctures near the apex of the right lower lobe yielded no fluid. Signs of a suppurative process in the left shoulder developed and the patient was transferred to the Surgical Division. An abscess was drained by an incision through the deltoid muscle. The pus contained streptococci. A septic temperature continued; the *x*-rays showed infiltration of much of the right lung, and signs of consolidation of the apex of the right upper lobe became more marked. A large subcutaneous abscess appeared behind and below the left shoulder and this was incised. At about 2.30 P.M on April 12, a needle was inserted in the upper portion of the left chest posteriorly in search of other collections of pus. A few minutes after this puncture the patient complained of a choking sensation and became dyspneic with profuse sweating. Physical signs of pneumothorax developed in the left chest and death from asphyxia occurred at 12.10 A.M. on April 13.

Autopsy by Dr. Schultz showed universal, thick, pleural adhesions over the right lung which was filled with innumerable tubercles. The apex of the left lung contained many tubercles and the overlying pleura was adherent. The rest of the lung was collapsed. The site of the puncture was not found, but was probably in or near the adherent pleura. There was osteomyelitis of the scapula and head of the humerus involving the shoulder joint. As in the first case, it was the almost complete disability of the unwounded lung which led to the fatal termination from pneumothorax.

The point which I would emphasize above all is that the most serious and sudden results of the occurrences of pneumothorax due to exploratory puncture follow injury, not of the diseased, but of the efficient lung, when, as in two of the cases reported, the diseased lung is so incapacitated by consolidation and adhesions as to be incapable of adjusting itself to the unusual task. In the presence of a chronic inflammatory pulmonary process we are thus confronted with a choice of evils. Puncture of the area of chronic consolidation may result in hemorrhage or serious accident from pleural reflexes; puncture of the opposite normal lung may cause fatal pneumothorax. It is true that puncture of a normal lung rarely causes pneumothorax, but when it does do so, under such circumstances as existed in the cases reported, it is equivalent to signing a death-warrant. With these facts before us the conclusion seems inevitable that in the presence of probable chronic inflammatory disease of the lung, exploratory puncture should be restricted to cases in which there are good reasons for suspecting the existence of purulent exudate which would require surgical treatment.

TUBERCULIN THERAPY IN SURGICAL TUBERCULOSIS.

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PART I. ARTICLES AND MONOGRAPHS.

Koch's Era. The use of specific therapy in tuberculosis began with the era established through the discovery of Koch's tuberculin. Koch[1] recommended the injection of old tuberculin after careful experimental work upon healthy and tuberculous guinea-pigs.

Among the first to experiment upon the same line were two investigators in this country, Trudeau[2] and de Schweinitz,[3] both of whom obtained results corroborating Koch's experimental work, and it is safe to say that since their time no more important and convincing experiments have been done along the same line. In reviewing the literature dealing with experimental active immunization for tuberculosis, one is impressed with the fact that experimental evidence in favor of injection of tuberculin is far from